THE RECOVERY KERNEL

Substance-Specific Navigation Series

HOW TO RECOVER FROM KETAMINE

Using the Recovery Kernel Framework

Ketamine · Special K · K · Vitamin K · Ket · Dissociative Anesthetic · Research Chemical Analogs

⚠ HARM REDUCTION RESOURCE — NOT MEDICAL ADVICE | Crisis: call 911 or text 988

NOTE: This guide addresses recreational ketamine use disorder and problematic use patterns.

Medically supervised ketamine therapy (Spravato / esketamine) is a different context addressed in Section 1.3.

A Note Before You Begin

Ketamine sits at one of the most paradoxical positions in modern pharmacology: it is simultaneously a Schedule III controlled substance with significant abuse and addiction potential, a veterinary anesthetic, a club drug, and an FDA-approved treatment for treatment-resistant depression (as esketamine / Spravato). This guide exists for people whose relationship with ketamine has moved from recreational, therapeutic, or experimental use into a pattern that is costing them more than it is giving them.

The Recovery Kernel framework applies to ketamine recovery with several features that distinguish it from every other guide in this series. First, the primary organ damage is not neurological — it is urological. Ketamine-associated uropathy (the bladder and urinary tract damage from heavy ketamine use) is a medical emergency that is entirely separate from the addiction itself, and it requires its own treatment track that the Recovery Kernel framework does not replace. Second, the psychological dependence often exceeds the physical dependence — ketamine does not produce the classic physical withdrawal syndrome that opioids, benzos, or alcohol do, but the compulsive use pattern can be as entrenched as any in this series. Third, the dissociative state that ketamine produces has a specific psychological function for many users — escape from psychological pain, trauma, or

a reality that feels unbearable — that must be addressed directly for recovery to be durable.

And fourth: the same molecule that is destroying some people's bladders is being used to treat other people's suicidal ideation in supervised clinical settings. This complexity is held throughout this guide without trying to resolve it into a simpler story than it is.

Section 0 — Immediate Safety: Read This First

Acute Ketamine Overdose — Recognition and Response

THE CENTURION — Ketamine Overdose Is Primarily a Respiratory and Aspiration Emergency

Ketamine does not produce the same respiratory depression as opioids, and it maintains airway reflexes better than most anesthetics. However, at high doses or in combination with other central nervous system depressants, it can produce:

- Complete dissociation and unresponsiveness — the k-hole at clinical severity
- Vomiting while unable to protect airway — the primary aspiration risk
- Respiratory depression — particularly when combined with opioids, alcohol, benzos, or GHB
- Cardiovascular stimulation — elevated heart rate and blood pressure that can be dangerous in people with cardiac conditions
- Psychosis — at high doses, ketamine can produce acute psychosis that is clinically indistinguishable from schizophrenia

⚠ IF SOMEONE IS UNCONSCIOUS AND UNRESPONSIVE AFTER KETAMINE USE: CALL 911 IMMEDIATELY. ROLL TO RECOVERY POSITION (ON SIDE) TO PREVENT ASPIRATION. STAY WITH THEM.

⚠ COMBINING KETAMINE WITH OPIOIDS, BENZODIAZEPINES, ALCOHOL, OR GHB DRAMATICALLY INCREASES OVERDOSE RISK. THERE IS NO ANTIDOTE FOR KETAMINE.

The K-Hole — When Dissociation Becomes an Emergency

The 'k-hole' is the colloquial name for the state of complete dissociation that occurs at high ketamine doses — a profound disconnection from body, identity, time, and reality that can range from terrifying to mystical depending on context, dose, and set and setting. At recreational doses in non-medical contexts, a k-hole is a medical emergency because:

- The person cannot respond to their environment or protect themselves
- Vomiting while in k-hole can cause aspiration pneumonia or death
- Panic and extreme physical agitation can cause injury
- The person cannot communicate distress or summon help

Response to k-hole: do not leave the person alone. Place in recovery position. Clear the immediate area of hazards. Monitor breathing. If breathing becomes irregular or stops, call 911. Do not give other substances to 'bring them out of it.' Wait. They will emerge. The duration is typically 30–90 minutes for a full k-hole at recreational doses.

The Bladder — The Emergency That Cannot Wait

⚠ IF YOU HAVE ANY OF THE FOLLOWING SYMPTOMS, SEE A UROLOGIST OR EMERGENCY PHYSICIAN NOW — DO NOT WAIT FOR RECOVERY TO ADDRESS THIS:

- Severe urinary frequency — needing to urinate every few minutes
- Severe urgency — inability to delay urination
- Pain in the lower abdomen, pelvis, or flanks
- Blood in urine
- Urinary incontinence — loss of bladder control
- Upper urinary tract pain (kidneys, sides of abdomen) — indicates possible kidney involvement

Ketamine-associated uropathy (KAU) can progress from mild irritative symptoms to complete bladder contracture requiring surgical removal. The only way to halt this progression is to stop using ketamine. But some damage requires active medical treatment regardless of cessation. Section 1.4 covers this in detail. If you have bladder symptoms, urology consultation is the first priority — before the supplement stack, before the protocol navigation, before anything else.

Section 1 — What Ketamine Did to Your Brain and Body

THE CENTURION — The Counterfeit Sacred — Simulating the Highest Without Building the Foundation

The Recovery Kernel Compendium describes ketamine through a specific lens from its Centurian Paper on the chakra system and substances: ketamine simulates the highest center — the dissociative, ego-dissolving, transpersonal state associated with profound spiritual experience — while simultaneously destroying the most fundamental center. The compendium's exact language: 'The drug that simulates the highest center (third eye) simultaneously destroys the most fundamental center (root). This is the structural logic of the counterfeit sacred.'

This is not spiritual language imposed on a pharmacological reality. It is a description of what the neuroscience and the urology are showing: ketamine produces states that humans across cultures have sought for centuries through meditation, shamanic practice, and near-death experience — ego dissolution, unity, release from the tyranny of self-referential thought — and it does so by blocking NMDA receptors throughout the brain, including in the urinary tract, where the blocking of those same receptors produces progressive, irreversible tissue damage.

Understanding this dual reality — the genuine psychological value of the dissociative state AND the genuine physical destruction of chronic high-dose use — is the prerequisite for everything that follows.

1.1 — The NMDA Receptor — What Ketamine Blocked

THE CENTURION — The Brakes on Excitation — and the Engine of Learning

NMDA (N-methyl-D-aspartate) receptors are a subtype of glutamate receptors — part of the brain's primary excitatory system. They play a central role in:

- Synaptic plasticity — the ability to form and strengthen or weaken synaptic connections (long-term potentiation and depression)
- Learning and memory — NMDA receptors are required for the encoding of new memories
- Pain processing — particularly chronic and neuropathic pain
- Mood regulation — NMDA hypofunction is implicated in psychosis and dissociation
- Normal consciousness — the sense of being a continuous self embedded in a continuous reality

Ketamine is an NMDA receptor antagonist — it blocks these receptors, preventing glutamate from activating them. At sub-anesthetic doses, this produces: analgesia,

dissociation, euphoria, time distortion, and — at higher doses — the complete ego dissolution of the k-hole. At therapeutic doses in clinical settings, the same NMDA blocking produces rapid antidepressant effects, possibly by triggering compensatory glutamate surges that activate AMPA receptors and stimulate BDNF production.

1.2 — What Chronic Ketamine Use Did to the Brain

System affected	What chronic ketamine did	What recovery looks like
NMDA receptor system	Chronic NMDA blockade causes compensatory upregulation of NMDA receptors — the brain adds more receptors to maintain excitatory balance. When ketamine is removed, these upregulated receptors produce a glutamate rebound — the excitatory system surges, producing the anxiety, hyperarousal, and cognitive disruption of ketamine withdrawal.	NMDA receptor density normalizes over weeks to months with abstinence. The glutamate rebound anxiety is the primary acute withdrawal symptom. Magnesium (NMDA modulator) and NAC (glutamate homeostasis) are the primary early supplements.
Dopamine system	Ketamine produces indirect dopamine release in mesolimbic circuits and modulates dopamine synthesis through NMDA-related pathways. Chronic use produces the familiar dopamine downregulation of addiction — anhedonia, motivational deficit, reward circuit blunting.	Dopamine reconstruction follows the same principles as other substances in this series: precursor support (tyrosine), BDNF-driven neuroplasticity through exercise, natural reward architecture. The dopamine recovery in ketamine addiction is generally faster than in stimulant addiction because the primary mechanism is NMDA modulation rather than direct dopamine flooding.
Prefrontal cortex and cognitive function	NMDA receptors in the prefrontal cortex are particularly important for executive function, working memory, and reality testing. Chronic blockade produces measurable cognitive impairment: working memory deficits, attention problems, word-finding difficulty, and impaired reality testing that can persist months into recovery.	The compendium's CYP2B6 'dissociative logjam' — where poor metabolizers of ketamine experience prolonged cognitive impairment because the drug clears slowly — is the most extreme version of this. Cognitive recovery: BDNF (exercise), alpha-GPC, DHA, and the 40 Hz gamma protocol from the compendium's sonic architecture section.
Memory and	NMDA receptors in the	Memory function typically

hippocampal function	hippocampus are essential for memory encoding. Chronic ketamine use produces memory gaps, difficulty forming new memories, and the distinctive 'k-fuzz' — the post-use cognitive haze that can last hours to days and, with chronic use, begins to persist between sessions.	recovers substantially over 3–6 months of abstinence. Lion's Mane (NGF), DHA, and vigorous exercise drive the hippocampal neuroplasticity that restores memory encoding capacity.
Psychological dissociation as a learned state	Ketamine trains the brain to dissociate — to exit from present experience into a disconnected internal state. With chronic use, this dissociative tendency can persist even without the drug: depersonalization, derealization, and the sense of watching one's life from outside it become baseline states for some heavy users.	Grounding practices, somatic therapy, and mindfulness-based approaches that specifically build present-moment embodied awareness are the primary interventions for persistent dissociation in recovery. Ketamine recovery is the only guide in this series that explicitly requires somatic grounding work as a clinical priority.

1.3 — The Clinical Context: When Ketamine Is the Treatment

THE CENTURION — The Same Molecule — Radically Different Contexts

Esketamine (Spravato), the S-enantiomer of ketamine administered as a nasal spray, received FDA approval in 2019 for treatment-resistant depression and in 2020 for major depressive disorder with acute suicidal ideation. Intravenous ketamine infusions are increasingly used off-label for the same conditions through licensed infusion clinics.

This guide is not for people using ketamine in medically supervised therapeutic contexts with proper integration support. The compendium notes: 'The therapeutic dose exception: clinically administered ketamine at appropriate doses, with integration support, is a different intervention — the clinical model provides the container the recreational use lacks.'

However, three situations exist where therapeutic ketamine and this guide intersect:

- Therapeutic ketamine use that has escalated into self-administration, dose escalation, or use outside the prescribed clinical context — this is now recreational use disorder regardless of how it began.
- People who are in ketamine therapy for depression and are also recovering from recreational ketamine use disorder — the therapeutic use must be managed in full coordination with both the ketamine clinic and the recovery support system.

- People considering ketamine therapy for treatment-resistant depression who also have a history of recreational ketamine use — this requires specialist consultation before therapeutic use begins, as prior high-dose use may have altered NMDA receptor architecture in ways that affect therapeutic response and risk.

1.4 — Ketamine-Associated Uropathy: The Organ Damage That Cannot Be Ignored

⚠ KETAMINE-ASSOCIATED UROPATHY IS A PROGRESSIVE AND POTENTIALLY IRREVERSIBLE CONDITION. THE ONLY WAY TO HALT PROGRESSION IS TO STOP USING KETAMINE. SOME DAMAGE REQUIRES ACTIVE MEDICAL TREATMENT EVEN AFTER CESSATION.

Ketamine-associated uropathy (KAU) is one of the most severe and distinctive complications of heavy recreational ketamine use. It was first documented in clinical literature in 2007 and has become one of the defining medical risks of the current recreational ketamine landscape.

Stage	What is happening	Medical intervention required
Early — irritative symptoms	NMDA receptor blockade in urothelial cells disrupts normal bladder cell function. Inflammation of the urothelium (bladder lining). Urinary frequency, urgency, dysuria (painful urination).	Urology consultation. Cessation of ketamine use. Anti-inflammatory treatment. Most early-stage damage is reversible with cessation.
Intermediate — structural changes	Progressive fibrosis and inflammation of the bladder wall. Bladder capacity reduces. Upper urinary tract involvement may begin (ureters, kidneys). Hydronephrosis (kidney swelling from blocked drainage) possible.	Urgent urology consultation. Cystoscopy for assessment. Possible hydrodistension. Renal function tests. Cessation is non-negotiable at this stage — continued use causes rapid progression.
Severe — end-stage uropathy	Severe bladder contracture — the bladder shrinks to a fraction of normal capacity. This is the stage the compendium describes: 'ketamine cystitis — the scarring and contraction of the bladder that, at high doses, requires surgical removal.'	Specialist urology input. Possible cystectomy (bladder removal) and urinary diversion. This is irreversible. No recovery protocol addresses this — it is a consequence that requires surgical management. Cessation at earlier stages is the only prevention.
Biliary tract involvement	Less common but documented: ketamine can cause cholangiopathy — sclerosing cholangitis (inflammation and scarring of bile ducts). Presents as right upper quadrant pain,	Hepatology consultation. Liver function tests. Ultrasound of biliary tree. Cessation mandatory. Progression can lead to liver failure in severe cases.

	elevated liver enzymes, jaundice.	

If you have any urological symptoms, the first appointment you make is with a urologist — not with an addiction medicine specialist, not with a therapist. The bladder damage is the medical emergency. The addiction framework supports everything that follows. But the urology comes first.

Section 2 — Your Genomic Blueprint

Ketamine recovery has a specific genomic profile centered on two genes that the Recovery Kernel Compendium names explicitly in its practitioner reference: CYP2B6 (the 'Dissociative Logjam Risk' gene) and COMT (determining the specific character of the dissociative state's appeal). Understanding your profile also helps predict bladder damage progression risk and cognitive recovery timeline.

The DNA Testing Panel for Ketamine Recovery

Gene	What it controls	Key variant	Ketamine recovery relevance
CYP2B6	Primary metabolizer of ketamine — converts ketamine to norketamine in the liver	*6/*6 or *18 (rs3745274) — Poor Metabolizer	THE most important ketamine-specific gene in the compendium. The practitioner reference explicitly labels poor CYP2B6 metabolizers as carrying 'Dissociative Logjam Risk' — ketamine clears dramatically more slowly, producing: higher peak plasma levels from the same dose (accelerating addiction and organ damage), prolonged dissociative effects (the k-hole lasts longer), extended cognitive impairment after use, and potentially faster bladder damage due to longer urothelial exposure to active ketamine. If you ever felt that ketamine hit you much harder or lasted much longer than it did for others — CYP2B6 poor metabolizer status may explain this.
CYP3A4	Secondary metabolizer of ketamine alongside CYP2B6	Various inducers and inhibitors	CYP3A4 inhibitors (grapefruit juice, clarithromycin, fluconazole) can dramatically increase ketamine plasma levels — relevant both for understanding past use patterns and for avoiding inadvertent exposure during recovery. CYP3A4 inducers (St. John's Wort, rifampin) accelerate clearance. CYP3A4 is also the primary metabolizer of many medications used in recovery support.
COMT	Dopamine, norepinephrine, and stress chemical	Val158Met (rs4680)	Determines the specific character of what ketamine was providing. Sage constitution (Met/Met, slow COMT): ketamine's dissociative escape from

	clearance speed		the anxious, overstimulated mind was what was sought — the k-hole as the complete cessation of the ruminating, worrying thought stream. Warrior constitution (Val/Val, fast COMT): ketamine's euphoric, boundary-dissolving social state was more prominent — ketamine as the drug that made everything feel possible and connected. These different profiles drive different natural addiction porting strategies in Section 6.
MTHFR	Methylation — neurotransmitter synthesis foundation	C677T (rs1801133), A1298C (rs1801131)	MTHFR variants reduce baseline serotonin and dopamine synthesis efficiency. Depression, cognitive fog, and the sense of baseline deficiency that made ketamine's antidepressant-like effects compelling are often partly explained by MTHFR-variant methylation impairment. 5-MTHF supplementation addresses this directly and may explain why some people found ketamine so uniquely effective at treating a depression that nothing else seemed to touch.
DRD2 / ANKK1	Dopamine D2 receptor density — Reward Deficiency Syndrome	Taq1A (rs1800497)	A1 allele (reduced D2 density) predicts higher addiction susceptibility across multiple substances including ketamine. The 'something missing' baseline that DRD2 deficit produces makes the profound fullness of the ketamine dissociative state particularly compelling — the RDS brain finding in the k-hole the completeness it cannot generate at baseline. Recovery must build robust reward architecture or the RDS pull will be chronic.
BDNF	Brain-derived neurotrophic factor — neuroplasticity and cognitive repair	Val66Met (rs6265)	Ketamine at therapeutic doses works partly by stimulating BDNF production — a key mechanism of its antidepressant effects. Chronic high-dose ketamine paradoxically suppresses BDNF in some contexts through glutamate system disruption. Val66Met carriers have reduced BDNF secretion and may need higher exercise intensity to generate equivalent BDNF-driven cognitive repair. The cognitive recovery from heavy ketamine use depends heavily

			on BDNF activation.
SLC6A4 / 5-HTTLPR	Serotonin transporter expression — anxiety and mood sensitivity	Short/short allele	Short/short carriers have higher baseline anxiety and depressive sensitivity. If ketamine was being used to manage depression or anxiety that felt unreachable by other means, SLC6A4 short/short status explains part of the biological pull. Serotonin system reconstruction through 5-MTHF, 5-HTP, and exercise is particularly important for this profile.

Phenotyping Without Test Results

If genetic testing is not yet available, ask yourself:

- Did ketamine affect you much more intensely or last much longer than it seemed to for others at the same dose? → Likely CYP2B6 poor metabolizer. This is clinically significant information — your accumulated ketamine exposure per gram used was higher than average.
- Was the primary appeal of ketamine the complete cessation of thought, the escape from anxious rumination, or the sense of being freed from the burden of consciousness? → Sage constitution (Met/Met COMT) + likely SLC6A4 involvement. Depression and anxiety are the underlying conditions requiring direct treatment.
- Was the primary appeal the euphoria, the social dissolution, the sense of infinite possibility and connection? → Warrior or mixed COMT + DRD2 deficit components. The natural addiction porting needs to build those states through other means.
- Did ketamine seem to treat depression that nothing else had touched? → MTHFR variant + possibly appropriate candidate for supervised therapeutic ketamine for depression, but only after recreational use is addressed and under specialist oversight.
- Do you have a family history of multiple addictions or a chronic sense of 'something missing'? → DRD2 deficit / RDS profile. Multiple natural dopamine sources essential.

Section 3 — Detox and Withdrawal

THE CENTURION — Ketamine Withdrawal Is Primarily Psychological — But That Does Not Make It Less Real

Ketamine withdrawal does not produce the seizure risk of alcohol and benzos, the life-threatening cardiovascular instability of stimulant acute toxicity, or the severe physical suffering of opioid withdrawal. For most users, it is not a medical emergency.

What ketamine withdrawal does produce is a specific constellation of psychological symptoms that can be severe enough to drive immediate relapse, and a glutamate rebound state that produces anxiety, cognitive disruption, and perceptual disturbances that are genuinely distressing. The absence of dramatic physical withdrawal does not mean the person can simply stop without support.

The Withdrawal Syndrome — What to Expect

Timeline	Biology	Symptoms	What to do
Hours 0–24 (onset)	NMDA receptors upregulated during chronic use now fire without ketamine's blocking effect — glutamate rebound begins; dopamine system beginning to register absence of ketamine's indirect activation	Anxiety increasing, restlessness, dysphoria, strong cravings, irritability, cognitive fog, possible perceptual disturbances (visual effects, feeling of unreality)	Protocol 01 (Shelter) immediately. Magnesium glycinate for the glutamate rebound anxiety. Reduce environmental stimulation — the upregulated NMDA system makes sensory input feel amplified and threatening. Do not use other substances to manage withdrawal — particularly avoid benzos unless prescribed, as cross-dependence can develop.
Days 1–5 (peak psychological withdrawal)	Glutamate rebound at maximum; dopamine system in deficit; serotonin disruption; cognitive disorganization peak; CYP2B6 poor metabolizers may experience extended symptoms	Depression (often severe), anxiety, intense drug cravings, insomnia, vivid dreams, cognitive fog, possible depersonalization and derealization, anhedonia, possible emergence of the underlying depression or trauma that	This is the highest relapse risk window. Do not be alone. Contact support structure. NAC for glutamate homeostasis. Begin 5-MTHF if not already — the depression emerging may be the underlying condition that ketamine was treating. Medical support if depression is severe.

		ketamine was masking	
Days 5–14 (stabilization)	Glutamate rebound subsiding; NMDA receptor density normalizing toward new baseline; dopamine system slowly recovering; but significant neurochemical instability remains	Depression and anxiety persisting but less acute; cognitive fog beginning to lift; sleep slowly normalizing; cravings becoming episodic rather than constant; emergence of genuine emotions that ketamine was suppressing	Full supplement protocol. Begin exercise — BDNF drives both cognitive recovery and mood repair simultaneously. The emotions that emerge (grief, anger, loneliness, depression) are real and were being suppressed by the dissociation. They are not evidence that recovery is wrong. They are evidence of the person becoming present to their own life.
Weeks 2–8 (early recovery)	Progressive NMDA receptor normalization; dopamine system rebuilding; serotonin system recovering; cognitive function improving; the dissociative tendency reducing	Genuine improvement with variability; cognitive clarity returning; emotional range widening; the 'realness' of experience returning; cravings decreasing in frequency and intensity	This is when the grounding work of Section 6 becomes the primary focus. The person who was dissociating from their life through ketamine is now learning to inhabit it. This is the most important psychological work of ketamine recovery.
Months 2–12	Significant NMDA system normalization; dopamine and serotonin baseline recovered; cognitive function largely restored; bladder and urinary tract beginning to heal if cessation maintained	Stable mood with ongoing improvement; cognitive clarity matching or exceeding pre-use baseline; genuine present-moment experience accessible; craving primarily contextual	The long game: identity reconstruction around presence rather than dissociation. Trauma processing if trauma was underlying the use. Purpose architecture. The natural addiction porting strategy fully operational.

The CYP2B6 Dissociative Logjam — Extended Withdrawal in Poor Metabolizers

THE CENTURION — If Ketamine Always Hit You Harder and Lasted Longer — This Section Is for You

Poor CYP2B6 metabolizers clear ketamine dramatically more slowly than normal metabolizers. The clinical implications for withdrawal:

- Withdrawal onset may be delayed — ketamine clears more slowly, so the NMDA rebound happens later than expected
- Withdrawal symptoms may be more severe and more prolonged — the accumulated NMDA receptor upregulation from longer drug exposure per gram used
- Cognitive impairment in early recovery may be more pronounced and last longer
- Bladder damage may be more severe for the same reported dose history — longer urothelial exposure to active ketamine

If you suspect CYP2B6 poor metabolizer status, inform your recovery physician and any specialist providing treatment for ketamine-related organ damage. The dose history may underestimate your true ketamine exposure burden.

Section 4 — Biological Reconstruction

Ketamine recovery requires reconstruction of three systems: the NMDA/glutamate system (the primary pharmacological target), the dopamine system (secondary damage), and the cognitive architecture (the prefrontal and hippocampal functions that chronic NMDA blockade impaired). The methylation foundation underlies all three. Additionally, if bladder damage is present, urological recovery runs as a parallel track.

4.1 — The Methylation Foundation

Same foundation as every guide in this series, with one ketamine-specific emphasis: if ketamine was being used partly for its antidepressant-like effects, addressing MTHFR-variant methylation impairment is the most important early intervention for managing the depression that will emerge in withdrawal.

Supplement	Dose	Timing	Ketamine-specific notes
5-MTHF (L-methylfolate)	400–1000mcg	Morning with food	If ketamine was treating depression that felt biological and unreachable by other means, 5-MTHF may be the most important single supplement in recovery. MTHFR-variant methylation impairment produces serotonin and dopamine deficits that ketamine's NMDA-blocking and BDNF-stimulating effects were partially compensating for. Addressing the underlying deficit directly may reduce the intensity of the underlying depression that drives relapse.
Methylcobalamin (B12)	1000–2000mcg	Morning, sublingual	Chronic ketamine use depletes B12 and disrupts methylation cycles. B12 is essential for myelin integrity — relevant for the neurological recovery from chronic NMDA disruption. Sublingual form for best absorption.
P5P (active B6)	25–50mg	Morning	Cofactor for dopamine and serotonin synthesis — the neurotransmitter systems secondarily disrupted by ketamine's NMDA mechanism.

			Essential alongside the dopamine precursor stack below.
Magnesium glycinate	400–600mg elemental	Split: morning and evening	THE most important acute supplement in ketamine withdrawal because of its NMDA receptor modulating properties. Magnesium is an endogenous NMDA channel blocker — it provides gentle buffering of the glutamate rebound that produces the acute withdrawal anxiety. Start on Day 1 of cessation.

4.2 — Glutamate / NMDA System Support

THE CENTURION — Managing the Rebound — and Supporting the Recovery

The glutamate rebound of ketamine withdrawal is the acute pharmacological crisis. The NMDA system's recovery over the following months is the structural rehabilitation. Both require specific support.

Supplement	Dose	Timing	Role in NMDA recovery
Magnesium glycinate (repeated — highest priority)	400–600mg elemental	Morning and evening	Magnesium is an endogenous NMDA receptor channel blocker — it does exactly what ketamine does (blocks NMDA) but gently, physiologically, and without producing dissociation or abuse potential. During the glutamate rebound of ketamine withdrawal, magnesium directly modulates the excitatory surge. Continue for months — it also supports NMDA receptor normalization during the recovery phase.
NAC (N-Acetyl Cysteine)	600–1800mg daily in divided doses	Morning and evening with food	Restores glutamate homeostasis in the nucleus accumbens through the cystine-glutamate antiporter system. Multiple clinical trials support NAC for craving reduction in substance use disorders — its glutamate-normalizing mechanism is directly relevant to ketamine recovery where glutamate dysregulation is the primary neurochemical pathology.

Taurine	1–2g daily	With meals	NMDA receptor modulator — taurine acts as an inhibitory neurotransmitter at glycine receptors and modulates NMDA receptor function. Reduces glutamate excitotoxicity. Supports the liver function relevant to CYP2B6 ketamine metabolism. Dual mechanism relevant to both the neurological and metabolic aspects of ketamine recovery.
L-Theanine	200–400mg	As needed for anxiety — 2–3x daily in acute phase	Partial NMDA receptor antagonist and GABA/glycine agonist — provides gentle glutamate-dampening and calming through mechanisms parallel to ketamine's but without dissociation or addiction liability. Specifically relevant for the anxiety component of ketamine withdrawal where the glutamate rebound is the primary driver.
Omega-3 (EPA + DHA)	3–4g daily	With food, morning	DHA is structurally incorporated into neuronal membranes including those containing NMDA receptors — supporting membrane health supports receptor function normalization. EPA reduces neuroinflammation from NMDA excitotoxicity during withdrawal. BDNF support through DHA is particularly relevant for ketamine recovery.

4.3 — Cognitive Recovery: The K-Fuzz Clears

THE CENTURION — The Mind That Was Blocked Is Coming Back Online

The cognitive impairment of heavy ketamine use — what users often call 'k-fuzz' or 'k-fog' — reflects genuine neurological disruption: NMDA receptor dysfunction in the prefrontal cortex and hippocampus impairs working memory, attention, word-finding, and the clarity of thought that makes engaging with reality feel worthwhile. For CYP2B6 poor metabolizers, this impairment may be more severe and more prolonged.

The cognitive recovery timeline is generally 3–12 months for most users, with accelerated recovery in those who maintain vigorous exercise throughout. The mechanism: BDNF production from exercise drives hippocampal neurogenesis and synaptic plasticity — the same processes that ketamine's antidepressant effects were stimulating artificially.

- Exercise (primary intervention): 30–60 minutes aerobic daily. BDNF increases of 200–400%. This drives cognitive recovery faster than any supplement. The cognitive fog lifts measurably faster in people who exercise vigorously throughout recovery. This is the single most important cognitive intervention.
- Alpha-GPC or CDP-Choline (250–500mg morning): acetylcholine support for memory and attention. Ketamine impairs cholinergic function through NMDA-related mechanisms. Choline supplementation supports the recovery of memory encoding capacity.
- Lion's Mane mushroom (500–1000mg daily): NGF stimulation driving neurite outgrowth and synaptic repair. Specifically supports the hippocampal plasticity most relevant to the memory deficits of ketamine use.
- DHA (1000mg+ daily): structural support for neuronal membranes containing NMDA receptors. Supports BDNF signaling. Anti-inflammatory effects on the neuroinflammation from NMDA excitotoxicity.
- The 40 Hz gamma protocol: the compendium's sonic architecture section specifically identifies 40 Hz binaural/isochronic tones for their potential role in supporting the brain's glymphatic clearing processes and neural repair. 30–60 minutes daily as a cognitive recovery adjunct. Consistent use over months — not a quick fix, but a supportive background intervention.
- Novel skill learning: challenging new skills drive BDNF in specific brain regions. An instrument, a language, a physical skill with a learning curve — anything that generates errors and requires attention to correct. The prefrontal cortex damage from ketamine responds to exactly the conditions that skill learning creates.

4.4 — Dopamine and Serotonin Reconstruction

Supplement	Dose	Timing	Notes
5-HTP	50–150mg	Evening	Serotonin precursor — critical if depression was underlying the ketamine use. The serotonin system disrupted by chronic ketamine use responds to 5-HTP. Evening dosing supports sleep architecture alongside serotonin recovery. Do not combine with SSRIs or SNRIs without physician guidance.
L-Tyrosine / NALT	500–1500mg	Morning, away from protein	Dopamine precursor for the anhedonia and motivational deficit of ketamine recovery. Start after Day 7 once eating regularly. Particularly important

			for Warrior COMT profiles where the dopaminergic aspects of the ketamine state were primary.
Rhodiola rosea	300–600mg	Morning	Adaptogen with dopamine and serotonin modulating properties; anti-fatigue; cortisol normalization. Particularly useful in weeks 2–8 when the post-ketamine depression and motivational deficit are prominent but the precursor stack alone is insufficient.
SAMe (if depression is severe and MTHFR-related)	400–800mg	Morning, away from food	Universal methyl donor for dopamine and serotonin synthesis. Specifically relevant if 5-MTHF alone is insufficient for the depression that ketamine was managing. Caution in bipolar disorder — can trigger mania. Start low, assess response carefully.

4.5 — The Urological Recovery Track

THE CENTURION — The Bladder Heals When Given the Chance — But It Needs Help

Urological recovery from ketamine-associated uropathy runs parallel to the neurological recovery protocol and requires its own medical track. The supplements below have been studied or have mechanistic rationale for urothelial and bladder recovery — they do not replace urological medical care.

⚠ ALL UROLOGICAL SUPPLEMENTS SHOULD BE DISCUSSED WITH YOUR UROLOGIST. SOME — PARTICULARLY HIGH-DOSE VITAMIN C — CAN ACIDIFY URINE AND WORSEN BLADDER IRRITATION IN SOME PATIENTS.

Supplement / intervention	Dose	Timing	Urological relevance
Hydration — plain water	3–4 liters daily	Throughout the day	The most important urological intervention after cessation. Diluting ketamine metabolites (particularly norketamine) in urine reduces their contact concentration with the urothelium. Consistent hydration is the primary harm reduction tool for early-stage KAU and the

			primary recovery support for healing urothelium.
Quercetin	500–1000mg daily	With food	Anti-inflammatory flavonoid with specific evidence in interstitial cystitis (a condition with some mechanistic overlap with KAU). Reduces urothelial inflammation and mast cell activation. Well-tolerated. Discuss with urologist as part of the conservative management protocol.
Marshmallow root (Althaea officinalis)	1–2g extract or 4–5g root	Daily, can be taken as tea	Demulcent — coats and soothes inflamed mucous membranes including the urothelium. Long traditional use in urinary tract irritation. May reduce the dysuria (painful urination) and urgency of early-to-intermediate KAU. Discuss with urologist.
D-Mannose	1–2g daily	With water	Supports urothelial surface integrity — the glycoprotein coating of the bladder lining that is disrupted by KAU. Also protects against secondary bacterial colonization of a damaged urothelium. Evidence in urinary tract health broadly.
PEA (Palmitoylethanolamide)	300–600mg daily	With food	Endogenous anti-inflammatory fatty acid amide that reduces mast cell activation and neurogenic inflammation — both of which contribute to bladder pain and irritation in KAU. Evidence in interstitial cystitis and pelvic pain conditions. Does not interact with bladder pH.

4.6 — Sleep Reconstruction

Ketamine disrupts sleep architecture through multiple mechanisms: NMDA receptor disruption affects sleep regulation, the dopamine and serotonin disruption impairs sleep quality, and the psychological dependence often involved using ketamine specifically for sleep (the dissociative state as a substitute for genuine sleep). The vivid, disturbing dreams that often accompany early ketamine recovery are REM rebound — the brain reclaiming the restorative sleep stages that ketamine was suppressing.

- Melatonin: 0.5–1mg sublingual, 30–60 minutes before bed. Low dose. High doses disrupt circadian rhythm.
- Magnesium glycinate: 400mg elemental, evening. NMDA modulation, anxiety reduction, cortisol buffering — all support sleep onset.
- Glycine: 3–5g, 30 minutes before bed. NMDA co-agonist at inhibitory doses; reduces core temperature for sleep onset; improves sleep architecture.
- L-Theanine: 200–400mg evening. Alpha-wave promotion; reduces the glutamate-rebound anxiety that prevents sleep in early ketamine recovery.
- 5-HTP: 50–100mg evening. Serotonin → melatonin synthesis support. Important if depression-related insomnia is present.

Also execute Protocol 17 (Sleep Architecture) from the main Compendium in full.

4.7 — Peptide Support Layer

Physician discussion required — research compounds.

Peptide	Mechanism in ketamine recovery	Timing	Notes
BPC-157	Glutamate-dopamine system modulation; NMDA pathway effects; gut-brain axis repair; specific evidence for neuroprotection from NMDA excitotoxicity in animal models; potential urothelial healing properties	Day 1 onwards — daily	BPC-157 has demonstrated neuroprotective effects against NMDA excitotoxicity in research — directly relevant to the glutamate rebound of ketamine withdrawal. Its gut-brain axis repair function addresses the dysbiosis that ketamine use produces. The urothelial healing hypothesis — BPC-157's tissue repair properties extending to bladder epithelium — is mechanistically plausible though clinical evidence in KAU specifically is limited. Discuss the bladder application with your urologist.
Semax	BDNF upregulation; cognitive enhancement; dopamine modulation; prefrontal cortex support — directly targeting the cognitive impairment of ketamine use	Weeks 1 onwards — particularly months 1–6 for cognitive recovery	Semax's BDNF-stimulating effects directly accelerate the cognitive recovery from ketamine-induced NMDA dysfunction. The prefrontal cortex impairment and memory deficits of heavy ketamine use respond to

			BDNF-driven neuroplasticity. This is the peptide most directly relevant to the 'k-fuzz' recovery. Intranasal.
Selank	Anxiolytic via GABA and serotonin modulation; reduces anxiety without dependence; stable non-dissociative calming	Weeks 1–8 — as needed for acute anxiety	For the anxiety of ketamine withdrawal and early recovery, Selank provides genuine anxiolytic effect without the dissociative mechanism that made ketamine appealing in the first place. This is important: the person recovering from ketamine-as-anxiety-management needs an anxiety management tool that does not work through dissociation. Selank provides anxiolysis through a completely different pathway. Intranasal.
Epitalon	Sleep architecture; pineal pathway; telomere protection; circadian normalization	Weeks 1–6; cycles as needed for persistent sleep disruption	Sleep disruption in ketamine recovery is both neurochemical (NMDA disruption of sleep regulation) and behavioral (patterns built around ketamine-induced sedation). Epitalon supports the pineal pathway that ketamine was disrupting and helps re-establish genuine sleep architecture.

Section 5 — The Ketamine Craving — Understanding and Interrupting

THE CENTURION — The Craving Is Not for the Drug. It Is for the Exit It Provided.

Ketamine cravings have a specific quality that distinguishes them from stimulant cravings (which feel like hunger for more) or opioid cravings (which feel like pain demanding relief). Ketamine cravings tend to feel like: the overwhelming desire to leave. To exit from consciousness, from reality, from an emotional state that is too large to tolerate. The craving arrives when the present moment becomes unbearable — and ketamine's offer is the most efficient exit available.

Understanding this changes the interrupt protocol. You are not fighting a desire for a substance. You are meeting a need for exit from an overwhelming state — and providing an alternative exit that does not require dissociation.

The Ketamine Craving Types

Craving type	What it feels like	Immediate interrupt
Reality escape craving (most common)	The present moment is too much — too painful, too anxious, too real, too relentless. The mind reaches for the exit that has always been reliable. The craving is less about wanting the drug and more about wanting to not be here.	This is the grounding interrupt — the opposite of dissociation. Five things you can see. Four you can touch. Three you can hear. Two you can smell. One you can taste. Then 4-7-8 breathing. Then cold water on the face. You are not suppressing the overwhelming state — you are grounding it in physical reality rather than escaping from it.
Depression / anhedonia craving	The flatness is unbearable. Nothing feels interesting, worthwhile, or real without the drug. The ketamine state felt more alive than ordinary life. The craving is for aliveness that the recovering dopamine system cannot yet generate.	This is the dopamine deficit responding. Exercise is the fastest available dopamine intervention. Cold exposure. Music. Contact with someone whose presence generates genuine warmth. The natural reward architecture from Section 6 — execute the highest-intensity item available.
Curiosity / exploration craving	Ketamine's dissociative state had genuine depth — profound insights, ego dissolution, the	This is the invitation to build what Section 6 calls the genuine mystical infrastructure —

	feeling of accessing something larger than ordinary consciousness. The craving is for that space of expanded perception, not for the drug specifically.	meditation, breathwork, flow states — that accesses related neurological states without the pharmacology. The craving for genuine transcendence is not a pathology. It is pointing toward a real human need that can be met authentically.
Social / contextual craving	Specific music, specific friends, specific environments, specific times of night — the associations built around ketamine use fire a sudden intense craving that arrives without warning.	Environmental exit first — leave the context before doing anything else. Protocol 05 (environmental redesign) has addressed the most prominent cue environments, but novel cues will appear. Exit. Contact. Ground. The sequence does not change.

THE KETAMINE CRAVING INTERRUPT — 5-MINUTE PROTOCOL

1. IDENTIFY THE TYPE. Reality escape? Depression/anhedonia? Curiosity/exploration? Social/contextual? The type determines the primary response.
2. GROUND IMMEDIATELY. Five things you can see. Four you can physically touch. Three you can hear. Two you can smell. One you can taste. This is not a metaphor — it is prefrontal cortex activation and present-moment anchoring that interrupts the dissociative 'exit' the craving is seeking.
3. EXECUTE 4-7-8 BREATHING. Four full cycles. The parasympathetic response reduces the overwhelm that made the exit feel necessary.
4. COLD WATER ON FACE AND WRISTS. The dive reflex activates within seconds — immediate parasympathetic shift. This is the fastest available grounding intervention outside of the breath.
5. MAKE CONTACT. One text or call. The isolation of dissociation is part of the pattern ketamine created. Contact is its structural antidote.
6. EXECUTE YOUR NATURAL STATE. From Section 6: movement, breathwork, meditation, cold plunge, creative work. You are not suppressing the need the craving represents. You are meeting it differently.
7. DOCUMENT. Trigger, type, intensity (0-10), action, outcome. The ketamine craving pattern documented over weeks reveals the specific internal states and external contexts that are driving the difficulty. This is diagnostic information for the therapy and for the natural addiction architecture.

Section 6 — Porting the Addiction: Genuine Access to What Ketamine Faked

THE CENTURION — Centurian Paper C-28 — The Counterfeit Sacred Becomes the Real Thing

The compendium describes ketamine as the counterfeit sacred: it simulates states that human beings have sought through contemplative practice, ritual, and spiritual discipline for thousands of years. The ego dissolution of the k-hole maps precisely onto what contemplative traditions describe as the dissolution of the small self. The expanded awareness of sub-anesthetic doses maps onto what meditators in deep practice report. The sense of connection with something larger than ordinary consciousness is not unique to ketamine — it is a neurological state that the human system was designed to access.

The difference between the ketamine version and the authentic version is exactly what the compendium names: the ketamine version provides the state without requiring the development of the architecture that makes the state sustainable, integrable, and genuinely transformative. The contemplative version requires years of practice and genuine transformation of the person who inhabits the state.

Ketamine recovery is unique in this series because the natural addiction architecture is not primarily about building intensity (stimulants), endorphin warmth (opioids), GABA calm (benzos/GHB), or anandamide flow (cannabis). It is about building genuine access to the transcendent states that ketamine was counterfeiting. This is the most demanding natural addiction architecture in the series — and potentially the most rewarding.

The Grounding Architecture — Presence Before Transcendence

THE CENTURION — You Cannot Integrate What You Have Not Embodied

The first task is the inverse of what ketamine was doing. Before building genuine access to expanded states, the person in ketamine recovery must build the capacity to inhabit ordinary states — to be present in their body, in the moment, in their actual life, without reaching for the exit.

This is somatic work. It is the work of coming home to the body that ketamine was helping escape from. For many people in ketamine recovery, the body is where the unprocessed material lives — the trauma, the grief, the anxiety, the pain that the dissociation was providing relief from. Recovery requires building the capacity to be with that material without being destroyed by it.

- Somatic therapy (Protocol 23 in the main Compendium, or external specialist): specifically for people whose ketamine use was managing trauma

or emotional pain stored in the body. EMDR, somatic experiencing, sensorimotor psychotherapy. These approaches work with the body's experience directly — the opposite of ketamine's body-exit mechanism.

- Grounding practice (5-4-3-2-1 sense anchoring): practiced daily until it is as automatic as the former ketamine use. The nervous system needs to build the neural pathway for returning to present-moment embodied awareness.
- Physical exercise specifically as embodiment: the goal is not fitness. It is presence in a body in motion. Running, swimming, martial arts, dancing — any physical activity that requires full attention to what the body is doing right now.
- Safe physical contact: for people whose ketamine use involved social contexts, rebuilding the capacity for physical presence with others without chemical disinhibition is part of the grounding work.

The Transcendence Architecture — The Real Thing

THE CENTURION — The States Ketamine Faked Can Be Accessed Genuinely

Practice	Neurological state produced	How to implement	For ketamine recovery specifically
Meditation — absorption / jhana states	Ego dissolution, temporal dissolution, unity experience — measurably similar to psychedelic and dissociative states on brain imaging	Dharma Recovery (Protocol 04). Begin with basic breath meditation. Build to longer sits. The full dissolution states may take years of daily practice — the journey is the point.	The compendium notes that Buddhist jhana states, Sufi hal, Hindu samadhi, and Christian mystical union all describe states that brain imaging maps onto NMDA-modulated dissociative states. The practices that cultivate these states build the prefrontal inhibition of default mode network activity that ketamine was producing chemically. The genuine version builds something the pharmaceutical version never did: integration. The expanded state becomes accessible and useful rather than merely escapist.
Holotropic or pranayama breathwork	Altered consciousness through CO2/O2 modulation — documented ego	Holotropic breathwork sessions (group or individual with facilitator). Wim Hof breathwork. Box breathing extended over 30+ minutes.	Breathwork accesses neurological states that overlap with NMDA modulation through hyperventilation-induced CO2 reduction, which affects NMDA receptor function. This is a legitimate pathway to the states ketamine was producing —

	dissolution, emotional release, and transpersonal experiences		available without pharmacology, with full consciousness and memory of the experience, and integrable into a life narrative.
Sensory deprivation / float therapy	Profound reduction in sensory input produces states of deep relaxation, time dissolution, and occasionally ego dissolution through a completely pharmacology-free mechanism	Float centers offer 60–90 minute sessions. Build to regular use — weekly if possible. The state deepens with practice.	Float therapy accesses the sensory reduction that the k-hole was providing through external stimulation removal rather than NMDA blockade. For the person who was using ketamine specifically for the sensory and cognitive quiet it produced, floatation therapy is the most direct pharmacology-free analog available. Some ketamine-recovery communities specifically use float therapy as a transition practice.
Peak experience through physical mastery	Flow state, runner's high, and peak performance states produce BDNF, endorphin, and dopamine co-release with measurable ego-boundary dissolution at their peak	Extreme sports, martial arts, endurance athletics, technical climbing — physical activities with genuine mastery demands that produce flow at high intensity.	For the Warrior constitution who found ketamine's social euphorics and boundary dissolution appealing, physical peak states provide the intensity and boundary dissolution through earned means. The mastery component adds what ketamine never provided: the genuine knowledge that you are capable.
Integration therapy and psychedelic-assisted therapy (if appropriate)	Professional therapeutic use of MDMA (for PTSD, in research settings), psilocybin (for depression and addiction, in	Only through licensed clinical research programs or licensed providers (for ketamine specifically). Not self-administration. Full therapeutic container required.	The compendium's Protocol 42 addresses psychedelic-assisted therapy specifically. For ketamine recovery where depression or trauma was underlying the use, supervised therapeutic psychedelics or therapeutic ketamine (in a clinical context with integration) may be appropriate under specialist guidance. These are

	clinical trials), or supervised ketamine (for treatment-resistant depression) — with full integration support		catalysts that open a window — the integration work determines the outcome.

Section 7 — Navigating the Recovery Kernel: Your Protocol Map

Phase	Protocols	Primary focus	Ketamine-specific notes
Immediate Medical (if bladder symptoms present)	Urology — not the Compendium	Urological assessment; cystoscopy; renal function; cessation	Bladder symptoms require urology before anything else in this framework. KAU progresses with continued use — cessation is the only way to halt it, and some damage requires active medical treatment regardless of cessation.
Biological Foundation (Days 1–14)	Protocol 01, Protocol 07, Protocol 17, Protocol 18	Glutamate rebound management (magnesium, NAC), methylation foundation, sleep, nutrition	Magnesium glycinate Day 1 — highest priority for the glutamate rebound. NAC Day 1. 5-MTHF Day 1 if depression is present. Protocol 01 (Shelter) for the acute dissociative anxiety of early withdrawal. Reduce environmental stimulation — the upregulated NMDA system makes everything feel amplified.
Genomic Mapping (Weeks 1–4)	Section 15 (Compendium), CYP2B6 testing	CYP2B6 (Dissociative Logjam Risk), COMT constitution, MTHFR, BDNF	CYP2B6 is the highest priority genomic test in ketamine recovery — it directly explains dose sensitivity, bladder damage severity, and cognitive recovery timeline. If you felt that ketamine always hit you harder than others, this test may be the most important medical information you can obtain.
Grounding Architecture (Weeks 1–8 — CRITICAL)	Protocol 01 (grounding practices), somatic therapy referral, Section 6.1 of this guide	5-4-3-2-1 grounding; somatic therapy; embodied exercise; physical presence practices	This is the phase that distinguishes ketamine recovery from every other guide in this series. Before transcendence architecture can be built, grounding architecture must be established. The person must be able to tolerate being present in their body and their life before building genuine access to expanded states.
Cognitive Recovery (Weeks 2–12)	Protocol 03 (exercise), Section 4.3 of this guide, novel skill learning	Exercise for BDNF; Alpha-GPC; Lion's Mane; 40 Hz protocol; new learning	The cognitive fog lifts measurably over months 1–6 in people who exercise vigorously. Track it — the return of cognitive clarity is one of the most motivating and reinforcing experiences of ketamine recovery.
Psychologica	Protocol 23,	Trauma processing if	The overwhelming states that

l and Trauma Work (Months 1–6 when stable)	external therapy (somatic, EMDR), Protocol 11	underlying ketamine use; depression treatment if biological; anxiety management	ketamine was providing exit from do not resolve with cessation. They require direct treatment. Somatic therapy, EMDR, CBT, or MBSR — depending on what the underlying condition is. This is not optional for durable ketamine recovery.
Transcendence Architecture (Months 2–12+)	Protocol 04 (Dharma Recovery), float therapy, breathwork, Protocol 42 (if appropriate)	Meditation building; breathwork practices; flow states; genuine access to expanded states	The building of the genuine mystical infrastructure — the practices that access the states ketamine was counterfeiting. This is the long game of ketamine recovery and it is also its greatest opportunity.
Identity and Meaning (Months 3–12+)	Protocol 20, Protocol 21, C-28	Identity as someone who inhabits reality rather than escaping it; purpose; the authentic version of what ketamine provided	For people who used ketamine partly for its spiritual or philosophical dimensions, the identity reconstruction includes integrating what was genuinely valuable in those experiences into a sober life. What was real about the insights? What was the expansion pointing toward? Protocol 20 and C-28 provide the framework.

Section 8 — The Medical Support Layer

Addressing the Underlying Depression

THE CENTURION — The Condition Ketamine Was Treating May Need Its Own Treatment

A significant proportion of people with ketamine use disorder began using partly because ketamine produced rapid and profound antidepressant effects that conventional treatments had not. The depression does not disappear with cessation — it returns, often more intensely, in the early weeks of recovery.

This depression must be treated directly. Options, in order of preference:

- 5-MTHF (400–1000mcg) + methylcobalamin: if MTHFR variants are suspected or confirmed, this is first-line. Addresses the specific methylation-deficit mechanism that makes some people's depression unresponsive to SSRIs. Try for 6–8 weeks before concluding insufficient.
- SSRIs or SNRIs: with CYP2D6/2C19 metabolizer status guiding selection. Discuss with psychiatrist. Note that SSRI response in people with ketamine use disorder is sometimes complicated by the NMDA system disruption — allow adequate trial periods.
- Supervised therapeutic ketamine (esketamine / Spravato): if the depression is genuinely treatment-resistant and other options have been exhausted, supervised ketamine therapy may be clinically appropriate even in the context of recovery from recreational ketamine use. This requires specialist addiction psychiatry input, not a general ketamine clinic.
- TMS (Transcranial Magnetic Stimulation): non-pharmacological treatment for depression with strong evidence in treatment-resistant cases. Modulates glutamate and NMDA pathways through electromagnetic stimulation — mechanistically relevant to ketamine recovery.

Managing Persistent Dissociation

Some people in ketamine recovery experience persistent depersonalization and derealization — feeling disconnected from themselves or their environment as a default state even without the drug. This can persist for months and is one of the most distressing features of heavy ketamine use.

Persistent dissociation in recovery is addressed through:

- Somatic therapy (primary): working with the body's experience directly to rebuild the sense of inhabiting a self. Sensorimotor psychotherapy, somatic experiencing, EMDR.

- Grounding practices (daily): 5-4-3-2-1 sensory anchoring. Cold water exposure. Physical contact with safe people.
- SSRI therapy: SSRIs have evidence in depersonalization disorder, which shares mechanisms with ketamine-induced persistent dissociation.
- Avoiding cannabis and other dissociative substances: cannabis use in ketamine recovery can significantly worsen depersonalization/derealization. Avoid.

Urological Medical Track

The urological recovery runs parallel to the addiction recovery and requires its own specialist team:

- Urology consultation: if any urological symptoms exist, this is the first medical appointment. Not addiction medicine. Not psychiatry. Urology.
- Cystoscopy: direct visualization of the bladder to assess the degree of structural damage and guide treatment planning.
- Renal function tests and ultrasound: to assess for upper urinary tract involvement.
- Hydrodistension: a urological procedure that stretches the bladder under anesthesia — can provide temporary symptom relief and allows assessment of bladder capacity.
- Intravesical therapies: direct bladder instillations of anti-inflammatory agents (hyaluronic acid, chondroitin sulfate) to support urothelial repair.
- Urology follow-up: 3-monthly for at least the first year of cessation to track recovery or progression.

A Final Word

THE CENTURION — The Exit Door Was Real. The Building You Were Escaping From Is Where the Life Is.

The compendium's description of ketamine as the counterfeit sacred is not a moral condemnation. It is a precise pharmacological and phenomenological observation: the states ketamine produces are real. The ego dissolution, the sense of expanded awareness, the release from the tyranny of the self-referential mind — these are genuine neurological events that map onto states human beings have sought and valued across every culture in recorded history.

What makes them counterfeit is not their character but their consequences. The path that provides them without the development — without the years of contemplative practice, without the gradual building of the psychological integration capacity, without the steady cultivation of the ability to remain present in ordinary states — also provides them without the fruit. The insight that cannot be integrated. The expansion that cannot be sustained. The peace that requires another dose. And, increasingly, a bladder that is slowly being destroyed by the molecule that was providing the transcendence.

Recovery from ketamine is not the abandonment of the states you were seeking. It is the discovery of the path that actually leads there.

The meditation hall is built for exactly what the k-hole was approximating. The float tank provides sensory dissolution without NMDA receptor damage. The breathwork session produces the ego boundary dissolution through means the body owns rather than means the pharmacy supplies. The flow state of physical mastery creates the sense of effortless, timeless absorption that sub-anesthetic ketamine was producing chemically.

The difference is that these paths build something. Each session leaves a residue — a slightly more developed capacity to inhabit the full range of human experience. A slightly more stable foundation for the next expansion. A slightly deeper integration of what the previous expansion revealed.

Ketamine was leaving a different residue. In the urothelium. In the prefrontal cortex. In the NMDA receptors that were trying to maintain the conditions for genuine consciousness.

The building you were escaping from is where the life is. The Recovery Kernel framework is the map for inhabiting it.

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Ketamine-associated uropathy: see your urologist before anything else.
Refer to the Recovery Kernel Complete Centurion Compendium for all referenced protocols.

Crisis: call or text 988 | Emergency: 911 | SAMHSA: 1-800-662-4357